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14. ABSTRACT Environmental heat stress can challenge the limits of human cardiovascular and temperature regulation, body fluid balance, and thus aerobic performance. This mini-review proposes that the cardiovascular adjustments accompanying high skin temperatures (T _{sk}), alone or in combination with high core body temperatures (T _c), provide a primary explanation for impaired aerobic exercise performance in warm-hot environments. The independent (T _{sk}) and combined (T _{sk} + T _c) effects of hyperthermia reduce maximal oxygen uptake (VO ₂ max), which leads to higher relative exercise intensity and an exponential decline in aerobic performance at any given exercise workload. Greater relative exercise intensity increases cardiovascular strain, which is a prominent mediator of rated perceived exertion. As a consequence, incremental or constant-rate exercise is more difficult to sustain (earlier fatigue) or requires a slowing of self-paced exercise to achieve a similar sensation of effort. It is proposed that high T _{sk} and T _c impair aerobic performance in tandem primarily through elevated cardiovascular strain, rather than a deterioration in central nervous system (CNS) function or skeletal muscle metabolism. Evaporative sweating is the principal means of heat loss in warm-hot environments where					
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Mechanisms of aerobic performance impairment with heat stress and dehydration

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HIGHLIGHTED TOPIC | *Mechanisms and Modulators of Temperature Regulation*

Mechanisms of aerobic performance impairment with heat stress and dehydration

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Cheuvront SN, Kenefick RW, Montain SJ, Sawka MN. Mechanisms of aerobic performance impairment with heat stress and dehydration. *J Appl Physiol* 109: 1989–1995, 2010. First published August 5, 2010; doi:10.1152/jappphysiol.00367.2010.—Environmental heat stress can challenge the limits of human cardiovascular and temperature regulation, body fluid balance, and thus aerobic performance. This minireview proposes that the cardiovascular adjustments accompanying high skin temperatures (T_{sk}), alone or in combination with high core body temperatures (T_c), provide a primary explanation for impaired aerobic exercise performance in warm-hot environments. The independent (T_{sk}) and combined ($T_{sk} + T_c$) effects of hyperthermia reduce maximal oxygen uptake ($\dot{V}O_{2max}$), which leads to higher relative exercise intensity and an exponential decline in aerobic performance at any given exercise workload. Greater relative exercise intensity increases cardiovascular strain, which is a prominent mediator of rated perceived exertion. As a consequence, incremental or constant-rate exercise is more difficult to sustain (earlier fatigue) or requires a slowing of self-paced exercise to achieve a similar sensation of effort. It is proposed that high T_{sk} and T_c impair aerobic performance in tandem primarily through elevated cardiovascular strain, rather than a deterioration in central nervous system (CNS) function or skeletal muscle metabolism. Evaporative sweating is the principal means of heat loss in warm-hot environments where sweat losses frequently exceed fluid intakes. When dehydration exceeds 3% of total body water (2% of body mass) then aerobic performance is consistently impaired independent and additive to heat stress. Dehydration augments hyperthermia and plasma volume reductions, which combine to accentuate cardiovascular strain and reduce $\dot{V}O_{2max}$. Importantly, the negative performance consequences of dehydration worsen as T_{sk} increases.

hypohydration; fluid balance; environment; critical core temperature; fatigue

HUMAN TEMPERATURE REGULATION processes maintain core body temperature (T_c) over a narrow functional range despite elevated metabolic rates and exposure to very hot environments (44). During submaximal exercise in warm-hot environments, humans limit exercise hyperthermia by balancing metabolic heat gain with dry and evaporative heat loss. Steady-state T_c is proportional to both absolute and relative metabolic rate within the prescriptive zone (57, 67, 87). Greater heat strain results when exercise intensity increases (17), clothing and equipment are donned (61, 63, 81), and when environmental heat stress becomes more severe (66). The combination of exercise and environmental heat gain also place high demand on evaporative cooling. If fluids are not adequately replaced, then significant dehydration (>3% total body water; >2% body mass) often occurs. The independent and combined effects of envi-

ronmental heat stress and dehydration can challenge the limits of human temperature regulation and aerobic performance. However, the putative mechanisms responsible for impaired aerobic performance remain debatable.

Physiologists have long been intrigued by the ability of humans to maintain adequate cardiac output and blood pressure to simultaneously dissipate body heat and sustain muscle force generation in hot environments. Although our understanding of the complex interplay among the physiological systems that govern these responses has evolved, the precise mechanistic underpinnings to explain impaired aerobic exercise performance in warm-hot weather remain unknown. Exercise-heat stress with or without dehydration may impair aerobic performance via one or more mechanisms related to central nervous system (CNS), skeletal muscle (metabolism), or cardiovascular system (blood flow) function (12, 14, 15, 27, 37, 70, 73, 74). Focus on a high T_c has been the major mechanism and unifying explanation for impaired aerobic performance in the heat (43, 68, 69, 73, 74, 76, 77), while the historical emphasis of cardiovascular strain accompanying high T_{sk} and skin blood

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flow (SkBF) (85) has only recently been reconsidered in detail (22, 37, 53). Unlike T_c , T_{sk} is drastically influenced by both environmental conditions and thermoregulatory responses. The potential for T_{sk} to fluctuate broadly has important implications for SkBF requirements, cardiovascular function and oxygen delivery (52, 85, 86), sensory input (30, 80), evaporative fluid loss (51), and other important performance-related factors common to the heat. In this minireview, we define a $T_{sk} > 35^\circ\text{C}$ as “high” (above the thermoneutral zone) (88) while acknowledging that T_{sk} warmed over a broad range will likely produce a continuum of effects (53, 88). We also recognize that T_c (and the $T_c - T_{sk}$ gradient) can alter the T_{sk} - SkBF relationship (6, 52, 85).

The purpose of this minireview is to evaluate the strength of the evidence supporting how heat stress impairs aerobic exercise performance in warm-hot weather. Potential CNS, muscle, and cardiovascular phenomena are examined. We provide a pragmatic explanation for impaired aerobic performance in the heat, both with and without dehydration, using well-defined physiological and behavioral concepts. Our main thesis is that high T_{sk} (or combination of high T_{sk} with high T_c) reduces $\dot{V}O_{2\text{max}}$ due to cardiovascular limitations that are exacerbated by dehydration. A shrinking cardiovascular reserve requires greater relative exercise intensity and sensation of effort for any given exercise power output. When the effort is protracted, an exponential decline in aerobic performance is observed. We suggest that the evidence for aerobic exercise performance impairments in the heat is better explained by cardiovascular (and oxygen delivery) limitations, rather than a deterioration in neuromuscular factors related to deep body temperature per se. While we identify the cardiovascular system as primary for limiting performance in the heat, we acknowledge that the CNS and skeletal muscle systems also provide important sensory input to the conscious brain and play a role in performance impairment.

EXERCISE PERFORMANCE IN THE HEAT

The well-documented impact of environmental heat stress on aerobic exercise “performance” has been evaluated using time to exhaustion (TTE) tests (incremental or constant work rate) and time trial (TT) tests (self-paced) in both laboratory and field settings. Galloway and Maughan (31) conducted one of the first studies to systematically compare the effects of graded heat stress on prolonged aerobic exercise performance. Using an ambient temperature range of $4\text{--}31^\circ\text{C}$, they demonstrated that TTE was nearly 42 min shorter (44%) in the warmest environment, relative to the study optimum (11°C). Similarly, MacDougall et al. (58) showed that TTE was shortened by 42 min (47%) when T_{sk} was increased from ~ 29 to 35°C using a water-perfused suit. The size of the heat-related performance impairment in these studies ($\sim 45\%$) is roughly two times larger than the typical variability in the measurement itself [coefficient of variation (CV) $\sim 25\%$] (45). The magnitude of the signal-to-noise ratio (~ 2.0) illustrates the strong negative effect of environmental heat stress on aerobic exercise performance.

In self-paced competitive running events, Ely et al. (23) found (Fig. 1) that marathon running performance slowed by $\sim 2\%$ (2–3 min) in elite competitors, while 3-h finishers slowed by $\sim 10\%$ (18 min) as wet bulb globe temperature (WBGT)

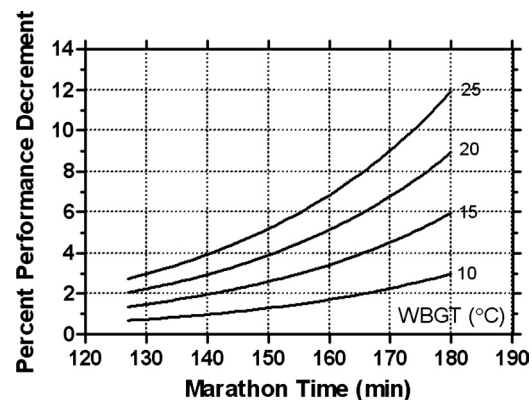


Fig. 1. Impact of weather on marathon running performance across a range of finishing times. As wet bulb globe temperature (WBGT) increases from 10 to 25°C [dry bulb temperature (T_{db}) = $8\text{--}22^\circ\text{C}$], elite competitors slow by $\sim 2\%$ (2–3 min), while 3-h finishers slow by almost 10% (18 min). WBGT, Reproduced from Ref. 23.

increased from 10 to 25°C (air temperature $8\text{--}22^\circ\text{C}$). Importantly, the smaller apparent effect magnitude (2 to 10%) is balanced by the fact that self-paced exercise is a more reliable test modality (CV 1–5%) (46), and thus the signal-to-noise ratio remains ≥ 2.0 . It is interesting to point out that in warmer weather, faster runners choose a slower, constant pace from the outset, while slower runners start at a faster desired pace only to decelerate (25). Tattersson et al. (94) observed similar findings in cyclists when comparing shorter (30 min), self-paced exercise in warm (32°C) vs. temperate (23°C) air temperatures. These latter studies indicate a change in behavior during self-paced performance tests due to heat stress and suggest that pacing strategy (28) may provide insight into how or why performance becomes impaired.

It is well established that heat stress degrades $\dot{V}O_{2\text{max}}$ (2, 34, 75, 93, 100) and that anything that reduces $\dot{V}O_{2\text{max}}$ can impair aerobic performance (3, 19). It is also well recognized that multiple physiological mechanisms can interact to influence aerobic performance outcomes. A shorter, self-paced aerobic test eliminates the potential for substrate depletion, marked heat storage, and dehydration common to more prolonged exercise in the heat. In order to test the hypothesis that higher T_{sk} alone might alter performance and pacing, Ely et al. (22) examined 15-min cycling TT performance in two environments (20 and 40°C), which produced T_{sk} of 31 and 36°C , similar to MacDougall et al. (58), while T_c remained near 38°C . Ely et al. (22) found that ratings of perceived exertion (RPE), heart rate (HR), and T_c were similar between self-paced trials. Total work performed at 40°C was 15–20% less than 20°C (with test CV $\sim 5\%$) similar to the performance decrement observed for the same task when ascending from sea level to 3,000-m altitude (60). Volunteers began the TT at the same pace in both environments, only to fall off pace at 40°C after the first 3 min of exercise (22). Collectively, TTE and TT studies demonstrate consistently that heat stress impairs aerobic exercise performance. Although cardiovascular (85, 86) and oxygen uptake adjustments to high T_{sk} (2, 34, 75, 93, 100) represent a plausible explanation linking perceived exertion (80) and behavioral changes (10) with impaired performance (22), mechanistic conclusions remain uncertain.

HYPERTHERMIA AND THE CENTRAL NERVOUS SYSTEM

Over the past 10 years, the most popular explanation for impaired exercise performance in a warm-hot environment has been a $\sim 40^{\circ}\text{C}$ "critical" T_c (43, 68, 69, 73, 74, 76, 77). The typical explanation is that this "critical" T_c threshold represents a safety brake for catastrophic hyperthermia (70, 74), or at least the precipice for a progressive downward performance trend (73). However, both of these interpretations can be challenged. First, a minimal lethal body temperature for humans is considerably higher (42°C) (8) and human CNS cells can tolerate blood temperatures in excess of 41°C without harm (20). Second, competitive runners (outside the laboratory) are often observed to achieve $T_c > 40^{\circ}\text{C}$ (even $> 41^{\circ}\text{C}$), without apparent sequelae, when T_{sk} was presumably low (9, 59, 84). Third, two recent studies could document no decline in running performance (velocity) during either short (8 km) or long (21 km) competitive running races despite $T_c \geq 40^{\circ}\text{C}$ (24, 56). Fourth, no study has clearly demonstrated an independent effect of a "critical" T_c , as confounding factors covary with T_c . Studies documenting an association between fatigue during dynamic, whole body exercise, and the attainment of a "critical" T_c were always performed under experimental conditions that produced high skin and muscle temperatures, narrow core-to-skin gradients, and substantial cardiovascular strain (43, 68, 69, 76), any one of which affords a tenable explanation for fatigue. Anticipatory models (97, 98) that focus on the rate of body heat storage appear untenable (24, 49), but mechanistic differences of theory are moot since both share logic that posits fatigue or its regulation (cortical or subcortical) based on avoidance of thermal injury.

Direct evidence of hyperthermia and degraded CNS function comes from associations between a "critical" T_c and altered brain wave (EEG), motor-neural output, and sensory changes consistent with fatigue (69, 76, 77). However, EEG alterations (69) may (78) or may not (83) be the consequence of reductions in brain blood flow, which ultimately represents a different (cardiovascular) mechanism (38, 79). It was recently demonstrated that the effects of heat stress on degrading neuromuscular function are progressive (95) and that as much as half of the neuromuscular fatigue effects attributed to a hot brain may be explained by hot muscles (96). It is not possible to say whether the remaining loss of efferent motor cortical output is the result of an unwilling or incapable participant, but it is well documented that the same exercise task in the heat is often perceived as more difficult or more uncomfortable than in temperate conditions (10, 30, 76). RPE involves a variety of integrated sensations that may converge to impact aerobic performance (10, 30, 80). Heat stress increases RPE (76). Anything that alters RPE may affect motivation driven motor-neural firing (11, 26) and behavior (10). Mundel and Jones (65) demonstrated that a menthol mouth rinse reduced RPE (compared with placebo) by 15% and improved TTE by 9% during exercise-heat stress. Similarly, Watson et al. (99) demonstrated that a 30% decrement in TT performance in the heat (compared with temperate control) was reduced to 19% with the same RPE when subjects were given a dopamine reuptake inhibitor. It would therefore appear that altered sensation can produce greater tolerance to exercise-heat stress at the cortical level. Importantly, however, the data of Watson et al. (99) illustrate that the remaining 19% performance decline in the heat (vs.

temperate) must be attributable to a larger, intact performance-limiting mechanism(s).

HYPERTHERMIA AND MUSCLE FUNCTION

Muscle function (mechanical and chemical) displays positive thermal dependence at the level of organismic performance (4). Muscle hyperthermia from exercise significantly increases the substrate flux through energy-producing biochemical pathways, improves phosphorylative efficiency, facilitates dissociation of oxygen from hemoglobin, reduces fluid viscosity, and enhances muscle contractility (4, 5, 7). In contrast, heating reduces time to exhaustion for sustained isometric contractions in small muscle groups (21). The importance of an isometric model to understand the relative CNS contribution to impaired force production in conjunction with a "critical" T_c was alluded to above. But the relevance of sustained isometric contraction of a small muscle toward aerobic performance in a dynamic whole body exercise model can be questioned on many fronts (4). For example, isometric contractions reduce skeletal muscle blood flow in proportion to the percent maximal contraction intensity (82), thus inducing very low oxygen contents and metabolite accumulation at fatigue. With the Q_{10} effect of heat stress (4), deoxygenation (hypoxia/dysoxia) and fatigue will occur more rapidly. Although quantifying deterioration in CNS function during whole body exercise may not be possible, it is well documented that disturbances in local (peripheral) muscle metabolism, muscle tension, or other factors can impact sense of effort (80). Muscle blood flow, a cardiovascular limitation, is preserved during submaximal aerobic exercise-heat stress (34, 71) and becomes reduced only as a secondary consequence of hypotension during severe heat stress (34). Although aerobic exercise in the heat can produce exhaustion long before muscle glycogen depletion is a contributing factor in endurance fatigue (27), the fact remains that substrate depletion is accelerated by heat stress (27, 50) at a time when oxidation rates of ingested carbohydrates are simultaneously reduced (50). Therefore, as submaximal aerobic exercise in the heat becomes protracted in length, limited fuel availability can contribute to impaired performance.

HYPERTHERMIA AND CARDIOVASCULAR FUNCTION

Exercise-heat stress demands blood flow to support energy metabolism (muscle blood flow), temperature regulation (SkBF), and CNS function (brain blood flow). When T_c and T_{sk} become elevated, there is a reflex increase in SkBF and cutaneous venous volume, while HR increases and cardiac filling and stroke volume (SV) decline (6, 52, 85, 86). When motivation is high and sensory cues ignored (10), a hierarchy is observed near maximal exercise intensities whereby muscle blood flow and arterial pressure are maintained at the expense of SkBF (37), which reaches a plateau near a T_{sk} of 38°C (6). Under circumstances in which considerable blood is displaced to the skin, blood flow and oxygen delivery to the muscles, and possibly brain (38, 78, 83), become compromised despite maintenance of leg vascular conductance (34). Table 1 illustrates that when T_{sk} is high, a rising T_c maintains a more favorable $T_c - T_{sk}$ gradient for reducing whole body SkBF requirements for heat loss (54). Figure 2 plots the apparent T_c tolerated, at task completion (24, 59) or at exhaustion (43, 81),

Table 1. Estimated whole body skin blood flow requirements for prolonged, severe running exercise at different body core and skin temperatures

T _c , C	T _{sk} , C	Gradient, C	SkBF, l/min
38	30	8	1.1
38	34	4	2.2
38	36	2	4.4
39	36	3	3.0

Equation for whole body skin blood flow (SkBF) requirements for prolonged, severe running exercise is $Q_s = 1/C \times h/(T_c - T_{sk})$, where C is the specific heat of blood ($0.87 \text{ kcal} \cdot \text{C}^{-1} \cdot \text{l}^{-1}$), T_c is body core temperature; T_{sk} is skin temperature, h = heat production (kcal/min), and Q_s = skin blood flow (from Ref. 85), and where, for severe running exercise, net heat production (7.7 kcal/min) is estimated using 60 kg body mass and 325 m/min running velocity (approximate pace for men's world class 42-km foot race) after subtracting for work (20% efficiency) and 50% dry and evaporative heat losses. Adapted from Ref. 54.

with its associated T_{sk} and calculated SkBF requirements (85). Note that T_c tolerance is inversely related to whole body SkBF requirements, which suggests that a "critical" T_c may instead be a perfusion issue (muscle, brain) related to high cardiovascular strain. Indeed, high T_{sk} has been an important historical index for heat tolerance (47, 48). The convergence of T_c and T_{sk} can be a good predictor of exhaustion (81), although predictive capacity may depend on the precise temperature at which T_c and T_{sk} converge (72). Sawka et al. (91) report that, when T_{sk} is made high by protective clothing or a severe environmental heat load, exercise is ceased (voluntary or collapse) in 50% of young healthy subjects at a T_c of only 38.5°C . Thus, in high heat stress situations with high T_{sk} , exercise cessation will occur relatively quickly in the absence of very high T_c at light-to-moderate exercise intensities.

During prolonged, submaximal exercise in less severe warm-hot environments, muscle blood flow and blood pressure are generally maintained, and thus explanations for fatigue are often attributed to the approach or attainment of a "critical" T_c (37, 43). But since a large $\dot{V}O_{2\text{max}}$ is a prerequisite for success in sports where aerobic endurance is contested (3, 19), it seems plausible that the effects of heat stress on reducing $\dot{V}O_{2\text{max}}$ might better explain submaximal aerobic performance impair-

ments. High T_{sk} reduces $\dot{V}O_{2\text{max}}$ as a consequence of high SkBF, which displaces blood to the periphery and reduces cardiac filling, resulting in a reduced maximal cardiac output (CO_{max}) (and thus $\dot{V}O_{2\text{max}}$) (2, 34, 58, 75, 85, 86, 93, 100). For example, Arngrimsson et al. (2) demonstrated a stepwise reduction in $\dot{V}O_{2\text{max}}$ and an increase in relative exercise intensity across a range of ambient temperatures (25 to 45°C) that increased T_{sk} by almost 6°C in the presence of modest T_c elevations. Although T_c plays a minor role in modifying the T_{sk} -SkBF relationship when T_c is high (6, 52), a high T_c can also reduce cardiac filling by tachycardia when sympathetic activity is high or by direct temperature effects on intrinsic HR (15, 29, 41). The reduction in both CO_{max} and $\dot{V}O_{2\text{max}}$ result in a shrinking cardiovascular reserve, which is the primary limiting factor for aerobic exercise performance (3, 19). A greater relative exercise intensity and sensation of effort is required for any given exercise power output (3, 10, 19, 32, 80). As exercise becomes protracted, performance is reduced exponentially according to the formula $\log_{10}(t) = A \times (\text{load}/\dot{V}O_{2\text{max}}) + B$, where t is endurance time and A and B are the slope and intercept relating t to the $\dot{V}O_{2\text{max}}$ -specific relative workload (32). Incremental or constant-rate exercise would be more difficult to sustain (earlier fatigue) or would require a slowing of self-paced exercise to achieve a similar sensation of effort. While a variety of afferent physiological information can be linked to the conscious sense of effort, the prominent contribution of cardiopulmonary factors (HR, respiration, baroreceptors) to RPE during whole body exercise is widely acknowledged (80). Earlier fatigue or slowing of pace can therefore be explained by greater cardiovascular strain (85, 86), elevated relative exercise intensity (3, 19, 32), increased perceived exertion (80), and associated behavioral changes toward sensory optimum (10).

EXERCISE PERFORMANCE WHEN DEHYDRATED

It is established that total body water deficits (dehydration) $> 3\%$ ($>2\%$ body mass) consistently impair aerobic exercise performance (14, 89, 90, 92). Practically, this means that if a person initiates activity in a normal state of body hydration (euhy-

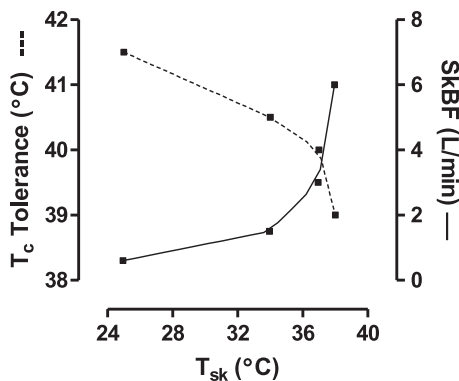


Fig. 2. Apparent core body temperature (T_c) tolerance during exercise-heat stress and calculated skin blood flow (SkBF) with associated skin temperature (T_{sk}). T_c and T_{sk} data pairs are from Refs. 24, 43, 59, 81. Heat production was calculated as in Table 1 starting from the actual metabolic data given in the original references. A point-to-point spline curve was generated with GraphPad to interpolate and visually smooth data connections for presentation only (no analytical model intended).

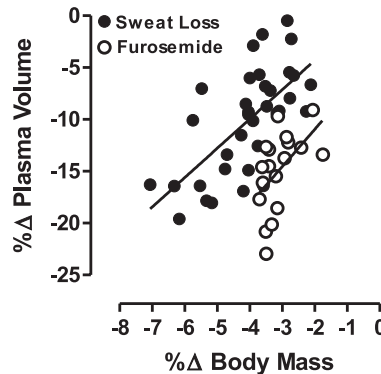


Fig. 3. Linear regression of plasma volume change (y) and body mass change (x) following sweat loss or diuretic dehydration. Original data are shown for 18 volunteers (54 data points) dehydrated twice by $4.1 \pm 1.5\%$ using 3–5 h of exercise-heat exposure (sweat; $n = 36$) and once by $3.1 \pm 0.5\%$ using 40–80 mg diuretic (furosemide; $n = 18$) on 3 separate occasions. Plasma volume contraction with furosemide ($y_F = -3.8 + 3.6x$) is double that for sweat ($y_S = 1.35 + 2.8x$) at any given level of dehydration. Plasma volume changes calculated according to Dill and Costill (18).

drated), dehydration-mediated performance decrements are restricted to activities lasting 1 h or longer as sustainable exercise sweating rates are typically <1.5 l/h. While dehydration can reduce muscle blood flow (35, 36) and alter skeletal muscle metabolism (27) during intense, exhaustive exercise in hot environments, intense exercise in hot environments is limited in duration and unlikely to be affected by dehydration if exercise is begun well hydrated. Headache, or even thirst (92), may also produce subjective feelings of fatigue or loss of vigor, but attempts to measure somatosensory gating of afferent neural signals in association with dehydration and performance are thus far inconclusive (64).

Hypertonicity is an unavoidable consequence of dehydration that helps mediate T_c elevation (89), which contributes to increased cardiovascular strain (15, 62). Dehydration by 2–4% body mass also produces an added challenge to meet whole body blood flow requirements by reducing plasma volume up to 10% (Fig. 3), which acts to reduce cardiac filling and SV (39, 40, 41, 42). Dehydration by 2–4% body mass generally reduces $\dot{V}O_{2\max}$, although the effects are larger in warmer environments (89). Just as plasma volume expansion by ~10% can improve $\dot{V}O_{2\max}$ by ~5% (16), Nybo et al. (75) demonstrated that 4% dehydration reduced blood volume by ~5% (plasma volume ~10%) and lowered $\dot{V}O_{2\max}$ by 6% at T_{sk} 31°C and by 16% when T_{sk} was raised to 36°C (water-perfused suit). As with heat stress alone, a reduced $\dot{V}O_{2\max}$ when dehydrated would make incremental or constant-rate exercise more difficult to sustain or require a slowing of self-paced exercise to achieve a similar sensation of effort.

Hyperthermia and plasma loss each account for ~50% of the dehydration-mediated decline in SV and CO_{\max} (15, 33), which we propose both can reduce $\dot{V}O_{2\max}$ and impair aerobic performance. This idea is also consistent with the observation that dehydration may impair exercise performance more under heat stress (14, 33, 41, 75, 89) compared with cold exposure (13, 41, 55). The strong modifying effect of T_{sk} on aerobic performance when dehydrated supports the proposed underlying SkBF demands and cardiovascular limitations on altering $\dot{V}O_{2\max}$, relative exercise intensity, and associated sensory cues. Indeed, Kenefick et al. (53) quantified the relationship between T_{sk} and aerobic performance degradation when dehydrated (4%) across a range of air temperatures (10–40°C) that incrementally elevated T_{sk} (but not T_c). During a short aerobic performance test that was identical to Ely et al. (22), and therefore not easily confounded by other factors, Kenefick et al. (53) reported a 1.6% decline in the total work performed for every 1°C increase in T_{sk} starting at a T_{sk} intercept of 29°C. At the same 4% level of dehydration, this amounted to a 3% impairment in aerobic TT performance at an ambient temperature of 10°C, increasing to 23% at ambient temperature of 40°C. Classic reports of larger aerobic performance reductions in temperate weather at more modest (1–2%) levels of dehydration (1) are likely the result of diuretic use, which doubles the plasma volume reduction observed from sweat loss (Fig. 3). A greater plasma volume reduction would presumably reduce $\dot{V}O_{2\max}$ and aerobic performance proportionally (89).

SUMMARY

This minireview evaluates mechanisms by which heat stress and dehydration impair aerobic exercise performance. The

physiological mechanism(s) that explain impaired aerobic exercise performance due to the independent and combined effects of heat stress and dehydration are highly circumstantial. The CNS and skeletal muscle systems provide important sensory input to the conscious brain, and there is evidence that each can play a role in impairing aerobic exercise performance during heat and/or dehydration stress. However, the demands and limitations on blood flow implicate a much larger performance contribution by the cardiovascular system. During severe heat stress conditions, physical exhaustion or collapse will occur across a continuum of T_c (91) and is most likely the result of the severe competition for blood flow, minimal $T_c - T_{sk}$ gradient, and inability to sustain required blood pressure (34, 37). During submaximal aerobic exercise in hot weather, performance is impaired by both high T_{sk} and T_c , which combine to reduce $\dot{V}O_{2\max}$ (2, 34, 75, 93, 100), and thus increase relative exercise intensity and sensation of effort at any given workload (TTE) (32), or result in a diminished workload (TT) to achieve a similar sensation of effort (22, 28, 94, 97). Significant dehydration (>2% body mass) takes time to develop, but when present it augments hyperthermia (89) and reduces plasma volume (Fig. 3), which independently impact physiology (15, 33) while accentuating the negative performance consequences of heat stress (53).

In this minireview, we provide a pragmatic explanation whereby high T_{sk} and T_c impair aerobic performance in tandem primarily through elevated cardiovascular strain, rather than deterioration in CNS function or skeletal muscle metabolism. Well-defined physiological and behavioral concepts support the plausibility of this explanation (3, 10, 19, 28, 30, 32, 44, 80). We emphasize the importance of T_{sk} (SkBF) in the context of aerobic exercise performance in warm-hot environments, with and without dehydration. We also revisit the important link between physiological strain and rated perceived exertion (10, 30, 80) to more fully explain how heat stress and dehydration act to impair aerobic exercise performance. Future research might consider the implications of this review when designing laboratory experiments to elucidate more precisely the mechanism(s) for impaired aerobic exercise performance during dehydration-heat stress.

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DISCLOSURES

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